as a possible risk factor for ventricular tachyarrhythmias. We compared QT dispersion in 120 patients with idiopathic dilated cardiomyopathy (DCM, 50 ± 12 years; EF: 31 ± 15%, range: 15–50%) and 60 age-matched controls. All 120 study patients with DCM were prospectively followed beginning from the time of QT dispersion measurement (baseline QT dispersion) until the end of follow-up in May 1995. Arrhythmic events during follow-up were defined as documented spontaneous sustained VT or VF, or sudden cardiac death, i.e. death within 1 hour after the onset of symptoms or unwitnessed death.

QT dispersion was defined as the difference between maximum and minimum QT intervals in the 12 lead ECG. QT intervals were measured in triplicate from 12-lead ECG, paper speed 50 mm/sec, using a high resolution computer digitizing table. QT intervals were measured from the beginning of the Q wave to the return of the T wave to the isoelectric line in each lead. In the presence of a U wave, the end of the T wave was defined as the intersection of the tangent to the repolarization slope with the isoelectric line.

Results: Baseline QT dispersion was 67 ± 10 ms in 120 patients with DCM compared to 40 ± 8 ms in 60 controls ($p < 0.01$). During 11 ± 6 months follow-up, 14 of 120 study patients with DCM (12%) had an arrhythmic event as defined above. Baseline QT dispersion was not significantly different between patients with arrhythmic events and patients without arrhythmic events (70 ± 12 vs. 65 ± 9 ms, $p > 0.05$).

Conclusions: 1) Compared to controls, QT dispersion intervals are significantly prolonged in patients with DCM. 2) The increased QT-dispersion in DCM appears to have little predictive value for the occurrence of major arrhythmic events during follow-up.

2:30

715-3 QTc Prolongation, but Not QTc Dispersion, Is a Significant Predicter for Death in a Normal Population

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Prolongation of QTc and increased QTc dispersion (QTc disp) have been found to predict ventricular arrhythmia and death in patients with ischemic heart disease. The aim of the investigation was to study the relation between QTc, QTc dispersion and death in a normal population.

Methods: From a population survey study performed in 1982, we examined 1951 persons. 12 lead ECGs were recorded at 25 mm/s. RR and QT intervals were measured with a digitizer tablet. QTc (ms, mean of 12 leads) was calculated using Bazett's formula and QTc dispersion as the maximal difference between QTc in any 2 leads in ms. The study population was aged 30, 40, 50 or 60 years. During the following 5 years 75 died.

Results: In a linear logistic regression model including QTc, QTc dispersion as well as age, gender, myocardial infarct, diabetes, hypertension, stroke, angina, claudicatio and medical therapy QTc ($p = 0.002$), but not QTc dispersion ($p = 0.09$) was an independent risk factor for 5 year mortality.

The table shows QTc and QTc dispersion (± standard deviation) for survivors and dead.

	Survivors	Dead	p
n	1876	75	
QTc	409 ± 26	418 ± 27	0.002*
QTc disp	62 ± 33	67 ± 32	0.09

Conclusion: Prolongation of QTc but not QTc dispersion is a highly significant risk factor for death in a normal population and is independent of age, gender, myocardial infarct, diabetes, hypertension, stroke, angina and medical therapy.

2:45

715-4 Exercise-Induced T-Wave Alternans as a Marker of High-Risk Patients With Hypertrophic Cardiomyopathy

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Previous studies have shown that the presence of subtle T-wave alternans (TWA) (alternating T-wave morphology from beat to beat) measured during atrial pacing and during exercise is predictive of spontaneous and inducible VT/VF in patients (pts) with ischemic heart disease. We tested the hypothesis that TWA measured during exercise could identify high-risk pts with hypertrophic cardiomyopathy (HCM). Of 14 pts with HCM, 7 pts were classified as high-risk for ventricular arrhythmic events (1 with sustained VT, 3 with increased fractionation of paced ventricular electrograms as seen in VF pts, and 3 with nonsustained VT and/or a family history of sudden death), and 7 pts without risk factors were classified as low-risk. All had no medication. There was no significant difference in age, sex and the degree of LV hypertrophy between the high-risk and low-risk pts. All pts were prospectively evaluated for TWA using the CH 2000 system with 7 multi-segment electrodes positioned in a Frank orthogonal (XYZ) configuration. In order to increase the heart rate (HR) up to 110/min, bicycle ergometer exercise was used. The alternans analysis was performed blind to clinical data. TWA was considered to be present if the alternans magnitude exceeded the previously published threshold of > 1.9 μV in X, Y, Z, V4 leads or the vector magnitude and was consistently present with the HR in excess of a pt-specific HR threshold. In 5 of 7 high-risk pts (71%), TWA was found at a HR of 95–110/min, whereas none of 7 low-risk pts (0%) had TWA ($p < 0.025$). Especially, all 4 pts with sustained VT or abnormal paced ventricular electrograms showed TWA. *Conclusions:* Pts with HCM who are at high risk for arrhythmic events often show TWA. Subtle TWA during exercise may be an excellent marker for arrhythmic risk in HCM pts.

3:00

715-5 QT Interval Dispersion Analysis in Patients With Acute Myocardial Infarction

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Qt interval dispersion (Δ Qt) may reflect left ventricular repolarization heterogeneity. Increased Δ Qt is associated with arrythmias post MI.This study investigates the effect of the infarct related artery reperfusion on Δ Qt.

Methods: 108 patients with acute MI submitted to the thrombolysis were divided in two groups: GA, with angiographic reperfusion (78 pts) and GB, without reperfusion (30 pts). The ECGs were analyzed at admission (pre) and after five days (post). The ECGs were amplified two fold in order to measure Qt interval on 12 leads. The Δ Qt was defined as the difference between the highest and lowest values obtained, and expressed in miliseconds.

	GA		GB	
	Δ Qt PRE	Δ Qt POST	Δ Qt PRE>	Δ Qt POST
Average (ms)	98 ± 40	66 ± 21	75.4 ± 22	94 ± 32

Δ Qt at the follow-up was significantly lower in patients with successful reperfusion (GA: $p < 0.001$). There was also a significant decrease in Δ Qt in patients with open arteries ($p < 0.001$) and a significant increase in patients without reperfusion ($p < 0.001$). Using decreased Δ Qt values as an index of reperfusion, the calculated sensitivity was 100% and specificity was 97.5% for the diagnosis of reperfusion.

In conclusion, Qt interval dispersion decreases in patients with successful thrombolysis, this could account for the smaller incidence of arrhythmias in these patients These findings also suggest that Δ Qt may be used as a marker of successful reperfusion.

3:15

715-6 QT Dispersion and Late Potentials in Hypertrophic Cardiomyopathy

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Recently, QT dispersion (QTd) has been proposed as a marker of susceptibility to non-sustained ventricular tachycardia (VT) in patients with hypertrophic cardiomyopathy (HC). Aim of this study was to detect a correlation, if any, between QTd, late potentials (LP) and VT. We performed standard ECG and signal-averaged ECG (40 Hz) in 29 patients with HC (21 men and 8 women, mean age 46 ± 15 years), with and without episodes of VT on 48 h Holter monitoring, and compared them to 50 sex and age matched healthy subjects. Patients were in sinus rhythm, without bundle branch block. None was taking antiarrhythmic drugs. LP were diagnosed when at least 2 of the following criteria were present: QRS > 114 ms, RMS-40 < 20 μV, and LAS > 38 ms. QT resulted significantly greater in patients than in controls (74 ± 31 vs 46 ± 12; $p < 0.001$) and in patients with VT than in those without it (109 ± 19 vs 53 ± 10; $p < 0.001$). Moreover, we analized the capability of LP and QTd ≥ 70 ms (corresponding to 2 SD above the mean calculated in our controls) to identify VT in HC.

	VT group (N = 13)	No VT group (N = 16)
QTd ≥ 70 ms	13/13 (100%)	0/16 (0%)
LP present	2/13 (15%)	2/16 (13%)